of the gall-bladder. If surgery is decided upon, several weeks or months of preparation will be required. In these cases also, preoperative preparation consists of rest in bed, a diet which includes high caloric fruit juices, fluids, digitalis, and careful and repeated checks on the function of the liver and the kidneys.

The patient who has severe diabetes in association with chronic disease of the gall-bladder should not be operated upon until every attempt has been made to get the diabetes under control. The diabetic patient, likewise, requires the closest coöperation between the surgeon and the internist, not only in preparing the patient for surgery, but also in directing his treatment following operation. The plan outlined for such a patient depends, of course, upon many factors, such as the age of the patient, the severity of the diabetes and the extent of the associated deficiency of the liver.

Before operation is performed on the patient with chronic disease of the gall-bladder and secondary anemia, the blood must be brought within normal limits, either by the use of one of the iron and liver compounds, or by blood transfusions. Transfusions are used much more frequently than they were in the past, and rightly so. During the time of such preparation, the store of glycogen in the liver can be increased by a high caloric diet rich in carbohydrates.

The patient who combines hyperthyroidism with chronic disease of the gall-bladder should have, by all means, the hyperthyroidism treated first; that is the rule and the practice in our clinic. After the hyperthyroidism has been controlled, usually by subtotal thyroidectomy after a period of preparation including rest in bed, the administration of Lugol's solution, phenobarbital and a high caloric diet, the patient will be able to undergo abdominal surgery with much less danger of a postoperative crisis. We have seen several such patients in our clinic, in some of whom the hyperthyroidism was not diagnosed until after entry to the hospital.

Patients with chronic disease of the gall-bladder associated with jaundice and marked hepatic damage, and all the complications which may be present, are very poor risks. I shall not enlarge upon this aspect further, as it will be discussed by other speakers on this program.

TESTS OF LIVER FUNCTION

It is impossible to urge too strongly the use of the different tests of liver function to determine damage to the liver. Any such test—Rose-Bengal, glucose, or any of the others—which will give adequate information, is well worth while. There is, however, a great need for a more adequate test of liver function. One or more such tests are routine in our clinic. If such a test shows marked damage to the liver, every attempt is made to improve the condition of the liver before operation. Time, rest, fluids, and glucose are very important to such a patient. The test should be repeated and surgery should be postponed until tests show the liver function to be within normal limits.

I am certain that we are all advising surgery earlier for our patients with gall-bladder disease, with better end-results. In connection with preoperative preparation, it likewise lowers operative risk. In most instances preparation of the patient can be carried on at home instead of in the hospital, thus reducing the cost of medical care.

OTHER PROCEDURES

We feel, as most medical men do, that it is perfectly possible for the patient to take an adequate amount of glucose by mouth, provided he is not nauseated. For example, he can easily take as much as 200 grams of sugar daily in fruit juice. We prefer to leave the intravenous method to be used following surgery.

COMMENT ON THE LITERATURE

A study of the literature indicates that the management of chronic disease of the gall-bladder has improved tremendously during the past ten or fifteen years, as shown by the reduced mortality rate. Our own figures show a reduction of almost 9 per cent. During the past nine years we have done some 317 cholecystectomies, with a mortality of one per cent plus, as contrasted with the previous period of nine years, when the mortality was around 10 per cent. The reasons for this are several. Better coöperation between the surgeon and the internist assures better preparation of the patient before surgery and selection of the proper time for the operation. Let me emphasize again that time is perhaps the greatest factor in the preparation of the poor-risk patient. There has been, as well, improvement in the anesthetic used and in the technical procedures. So simple a measure as the administration of glucose, though valuable, certainly should not receive all of the credit for the improvement in mortality statistics.

SUMMARY

Suggestions are made as to the preoperative preparation of the "bad risk" patient with chronic disease of the gall-bladder, to make operation less hazardous. Since there is so much individual variation in these cases, no hard-and-fast rule for such preparation can be laid down. Special points in regard to diet, vitamin and drug therapy are not included in this paper, as they are discussed by other speakers in this symposium.

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SINUS INFECTIONS: THEIR RELATIONSHIP TO RESPIRATORY DISEASE*

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SINUS infection is present in a great many respiratory diseases. It is often the most important etiologic factor.

Griffiths [1] reports a series of 5,000 cases in which the incidence of sinus infection was 385. Many of

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these cases showed secondary chest infections and persistent symptoms of intractable cough. Kerley [2] has reported the presence of demonstrable sinus disease in 133 cases out of 173, patients presenting themselves with acute respiratory infection. Kern and Donnelly [3] report as high as 62 per cent demonstrable sinus infection in asthmatic cases.

The above represents a rather large percentage of involvement of cases quoted. It is my belief that there is an even greater number of cases in which sinus infection is the responsible factor.

ANATOMICAL AND HISTOLOGIC CONSIDERATIONS

There is a normal connection between the nasal sinuses and the chest through a network of lymphatic vessels and the glandular system. This has been proven by various experiments; such as, the installation of dyes, carbon black, India ink, etc., into the sinuses of living animals. Their distribution follows a definite pathway; the antra and frontal sinuses draining into the upper and middle deep cervical glands, submaxillary glands, tonsils and bronchial glands. Main drainage of the antra and tonsils is into the glands of the hilum, and thence into the entire bronchial tree.

The histologic structure of lining of the sinus membranes is of such a nature that it becomes easily infected. The goblet cells and basement membrane undergo consecutive pathologic changes, with or without regeneration, depending upon the acuteness or chronicity of the disease, as has been demonstrated by Semenov,[4] and many others. Many prominent authorities make the statement that the normal histologic sinus membrane is unknown except in very small children.

Bacteriological studies by Turner and Loew,[5] Dochez [6] and others have shown that 85 per cent of nasal cultures of infants at birth are sterile; but that after the first feeding, multiple types of bacteria are found in the nose and nasal cavities. However, the lining membrane of the sinuses remains normally sterile until some condition intervenes which lowers the resistance of the membrane to infection. It has been shown that the greater majority of individuals have more or less persistent, ever-present pathologic changes in their sinus membranes.

PATHOLOGY

Although we are all familiar with the usual pathologic changes found in the nasal membranes in sinus disease, attention is invited to a few specific conditions found there. In subacute and chronic infection there is a destruction of cilia and goblet cells, to a greater or lesser extent, depending upon the severity and length of the infection. Goblet cells become smaller and fewer, and cilia become less or disappear. The basement membrane becomes thickened and shows an increase of connective tissue. Imbedded in this membrane are found colonies or inclusion nests of bacteria. These bacteria may be surrounded by cell products of a chronic inflammatory process; such as, cell débris and white blood cells. It is to these so-called nests or inclusion bodies that attention is directed.

The lymphatic glands are likewise subject to these chronic inflammatory changes and, of most importance, also have inclusion nests of bacteria present. In old cases these nests may have a giant cell nodular appearance similar to a tubercle. These nodes may be partially calcified, as is the tubercle in an old arrested case of pulmonary or glandular tuberculosis.

Cell débris may be present in the sinuses. The absence of pus is not of itself proof that there is no infection present. Polyps, mucoceles, and the various types of epithelioid tumor masses may be present. Small cysts may be found grossly or microscopically in the membrane of the sinuses. In all of these there are demonstrable pathogenic organisms present.

BACTERIOLOGY

Most all known bacteria are to be found in the sinuses of man. The ones of the most frequent occurrence are the various forms of streptococci, staphylococci, pneumococci, colon bacillus and influenza bacillus. In chronic infection there is a predominance of streptococci, and it is these bacteria that are responsible for most of the chest complications occurring with, or, as sequelae to, sinus infection.

Turner and Loew [7] have described a type of staphylococcus, termed chromogen 6, which they thought was the organism most frequently found in suppurative sinus disease. They found this group of bacteria to have an elective affinity for the mucous membrane of the upper respiratory tract and nasal cavities.

Meyersburg,[8] et al., describe as the causative organism the fusiform bacillus and spirillum of Vincent, found present in cases of suppurative chronic sinusitis and rhinitis caseosa.

Sewall [9] states that there is a filterable virus present in rhinosinusitis which activated the common pus-forming bacteria and sensitizes the individual.

ETIOLOGY

Just why one individual out of several that come in contact with the infectious organisms should develop respiratory disease presents the age-old, interesting question. Nearly everything in the realm of medical knowledge has been given as the cause. We recognize many predisposing etiologies such as climatic changes, environment, preëxisting diseases, diet, metabolic changes, and abnormal anatomic and physiologic conditions present.

Price [10] states that degenerative types in Pemhurst State School, because of abnormal vasomotor and endocrine disorders, were much more susceptible to respiratory infection than normal types.

Farmer [11] has pointed out that the large amount of histamine released during allergic attacks causes certain organic reactions in the walls of the blood vessels and in the smooth muscles, thus paving the way for infection.

Also, to be mentioned are the change in acid ion concentration and other biochemical changes that take place in the tissues of the infected individual.

The immediate cause of the respiratory infections is, of course, the pus-forming organism. It

has been stated by Sewall,[12] Carmody,[13] and many others, that recent colds are caused often by acute activation of old remote infections from residual germ-nests in chronically infected sinuses.

Just what is the agent that renders the individual acquiring a respiratory infection susceptible to the invading germs is a question that has not so far been answered satisfactorily, unless we accept the virus activator theory.

Recently there has been a great deal of work done in the study of tuberculosis. Briefly, investigations from many sources have reported that they have found within the tubercle of an individual, who has had or is suffering from tubercular infection, a substance that they have termed tuberculo-protein. This has a definite chemical structure and has been produced by pharmaceutical houses, and it is now available to the profession in the form of a purified protein derivative that is used for tuberculin tests. Tuberculo-protein has been isolated from the calcified nodes of individuals who have arrested cases of tuberculosis. It has been found in lesions anywhere which have been caused by tuberculosis, no matter whether arrested or not. All tubercular tissues contain protein bodies which are given off into the blood stream of the patient.

Persons who have inactive or active tuberculosis have an allergic reaction to tubercular protein. This may be due to the antigenic reaction caused by some of these proteins. Two water soluble proteins have been isolated from the tubercular bacillus, one acting like tuberculin. One other protein has been isolated that is devoid of tuberculin properties.

I would like to submit this purely speculative reasoning: that there are also bacterial proteins given off into the blood stream from inclusion nests and infected membranes containing other pathogenic organisms; that these proteins may act as sensitizing or activating agents; that these proteins may themselves be allergens or, if not true allergens, act as activating agents for other allergens; that the presence of these bacterial proteins in the blood-stream increases the susceptibility of the individual toward new respiratory infections or exacerbation of the old.

There is no definite bacteriologic or biochemic basis to support these contentions. There is, however, a clinical basis for some of this reasoning.

Patients do recover from chronic respiratory infection when the sinuses are drained or infected membranes are removed and autogenous vaccine administered. What exact rôle the vaccine plays is in question. We have learned that autogenous vaccines do contain antigenic substances which are many and complex in form.

Is the benefit from vaccine obtained because of the desensitization that can be attributed to the presence of these antigenic substances in the vaccine?

Little is known at present about the complex chemical structure of the streptococci. It has been determined, however, that some have a carbohydrate, a nucleoprotein that is antigenic and, as far as is known, some inactive elements. Also, in some streptococci the carbohydrate plus the protein is antigenic, and in some it is not.

It is interesting to question whether or not other protein or carbohydrate bodies from other bacterial sources do act as synergists or activators to the streptococci or cause production of antigenic substances by the germ.

DIAGNOSIS

There is no need to dwell at length upon the usual methods in use by competent rhinologists. Special care should be exercised in securing good x-ray pictures of suspected sinuses. The use of lipiodol is of great help in outlining the contour of the sinuses and demonstrating the filling defects found in the presence of a tumor mass or thickened membrane.

Diagnostic lavage plus culture of the contents will aid in identifying the offending organisms. In this connection I have had best success with a brain blood broth culture. Many varieties of streptococci do not grow well on ordinary media.

Bacterial skin tests are not of much value. The streptococci, with the exception of erthroprotein of scarlet fever, do not cause skin reactions usually.

Tuberculin skin tests are of value. A positive test may mean that there are tubercular foci present which are giving off specific allergens into the blood stream.

The history of onset, character of symptoms, amount, kind and frequency of discharge, etc., are of great value in determining the acuteness or chronicity of the disease.

If an allergic basis for the disease is suspected, the various food and other allergic tests should be carried out.

TREATMENT

In acute cases no surgery should be done until the infection has quieted down. Much damage may be done and many complications ensue because of hurried, inopportune surgery. Time must be given for nature to set up defensive barriers before undertaking surgery in the vast majority of cases. There are exceptions, as in cases of acute fulminating empyema of the frontal where drainage cannot be secured by the usual nonsurgical methods.

Sinus surgery in children should be rarely performed. I do not believe in tonsil and adenoid removal during the course of an acute infection. In chronic cases or subacute cases, the tonsils and adenoids are often the site of residual infection and should be completely removed.

It has been stated that people with normal noses do not contract sinus disease. I believe this is essentially correct. Nasal abnormalities should be corrected. It is my opinion that the submucous resection, properly done, is one of our most valuable operations. Many times a seemingly intractable respiratory infection is cleared up by securing proper drainage and areation of the nasal passages. To be of benefit a submucous resection should remove all the obstruction and not be a button-hole operation. It takes time and great care to do a successful submucous.

When the antra are chronically infected and have thickened membranes or tumor masses present, I believe in thorough removal of these membranes through a sublabial incision. Intranasal openings are not sufficient. In pansinusitis the transantral-ethmoid operation is the one of choice.

I dislike the term radical. We should consider sinus surgery in terms of other surgery. We do not speak of a radical appendectomy or cholecystectomy. When diseased tissue should be removed, it should not be termed a radical operation. Instead, it should be explained as a necessary operation.

I have had some small success in using an autogenous vaccine made from the culture of the tissue removed. The best results have been with vaccine made by the method described by Dimmit[14] for streptococci material. This vaccine presumably contains more antigenic factors than those made from agar cultures. It should be given in very small doses, always keeping below the reaction threshold. This is in accordance with the theory of desensitization. Reaction doses destroy the antiallergic factors developed in the system.

All cases of sinus disease in respiratory infection do not call for surgery. A large percentage of cases will respond to local treatment, tissue shrinking solutions, lavage by Proetz method, or otherwise.

We are all familiar with the necessity of proper climatic conditions, food, correction of general physical defects, etc.

If a given case has a positive tuberculin reaction, antiallergic treatment with "Old Tuberculin," using very small doses, is of benefit.

It is a mistake to assume that any vaccine or protein injection treatment will cure the sinus infection unless proper drainage has been obtained and foci of infection have been removed.

I have not had sufficient experience with the deep therapy treatment of sinus disease to be able to express an opinion as to its benefit. The rays are thought to cause formation and liberation of certain proteins into the system. These act as antigenic substances. This resembles the action of vaccines. How true this is, is a question at this time. Much remains to be done to increase our knowledge of the chemical structure and characteristics of bacterial and tissue proteins.

SUMMARY

Infections of the nasal accessory sinuses are important etiologic factors in respiratory disease. The pathway of extension is by means of the lymph and glandular systems. Are the bacterial proteins, known as allergens, activating agents? Treatment should include restoration of normal breathing space, removal of infected material and supportive measures.

CONCLUSION

The duration of respiratory disease can be shortened by treatment of the infections present in the nose and throat.

A conservative attitude should be held in regard to surgical procedures in children.

Surgery should not be attempted in acute fever stage of nasopharyngeal disease. Surgery, when performed, should be directed toward the removal of all infected tissue.

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PRIMARY REPAIR OF TENDONS*

STUDY OF END-RESULTS IN TWO HUNDRED AND SEVEN CASES

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THE increased incidence of injuries involving severed tendons, in both industrial and automobile accidents, justifies a survey of the whole subject of primary suture of tendons. A second and more important justification is the fact that almost all severed tendons are seen first and usually sutured by general surgeons.

A review of the literature impresses one with the paucity of articles on primary suture, and with the fact that most writers approve of primary suture in only strictly limited cases. Thus, Koch says: "Immediate repair of divided tendons and

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